

11:45

CARDIAC ARRHYTHMIA SUPPRESSION TRIAL (CAST): BASELINE PREDICTORS OF HIGHEST MORTALITY

Edward V. Platia, Richard W. Henthorn, Yudi Pawitan, Thomas A. Buckingham, Mark D. Carlson, Jeffrey L. Anderson, Peter E. Carson, and CAST Investigators  
Washington Hospital Center, Washington, D.C.

As part of the CAST, we analyzed clinical and laboratory baseline variables, and found that the 755 patients (pts) treated with flecainide and encainide (FLEC/ENC) and the 743 pts receiving placebo were similar. Our objective was to determine which variables reflected the greatest disparity in mortality between pts treated with FLEC/ENC and those treated with placebo. We calculated relative risk (RR) of total mortality for each variable, at an average of 10 month followup. For each of the variables examined, the total mortality was greater for FLEC/ENC compared to placebo pts. However, there were several univariate predictors of particularly high RR: (all  $p < .0004$ ): The highest RRs were observed in pts with a non-Q wave MI (RR=9.1), history of prior MI (RR=3.1) or angina (RR=2.8), and heart rate  $>74/\text{min}$  (RR=3.0). The only significant interaction was found between FLEC/ENC and Q wave abnormality ( $p=.03$ ). The significance is nominal because it is not adjusted for multiple comparison.

In conclusion, although FLEC/ENC treatment was associated with higher mortality with respect to all baseline variables measured, the risk was greatest in pts with non-Q wave MI. This supports the hypothesis that a possible interaction between potentially jeopardized myocardium and FLEC/ENC was important in increasing mortality in the CAST.

Wednesday, March 6, 1991

10:30AM-12:00NOON, Room 264, West Concourse  
Factors Associated with Progression or Regression of Coronary Artery Disease

10:30

INCREASED POTENTIAL FOR REGRESSION OF POST-PTCA RESTENOSIS USING INTENSIVE LIPID-ALTERING THERAPY: COMPARISON WITH MATCHED NON-PTCA LESIONS

Zhao X-Q MD, Flygenring BP MD, Stewart DK MD, Albers JJ PhD, Bisson BD MPH, Bardsley JL, Brown BG MD PhD  
Cardiology Division, University of Washington, Seattle, Washington

To determine the potential for "late" regression of lesions  $\geq 6$  months post-PTCA, we studied 18 such lesions in 16 patients with baseline catheterization  $7 \pm 4$  months post-PTCA and at follow-up cath 2.5 yrs later. These patients were among those participating in the FATS Trial. All had age  $\leq 62$ , apoB  $\geq 125$  mg/dl, positive family history, and established coronary disease. All had been randomly assigned to L+C: Lovastatin 20 mg bid plus colestipol; N+C: Niacin 1 gm qid plus colestipol; or CONV: placebos (or colestipol 10 mg tid if LDLc was elevated). Quantitative coronary arteriography demonstrated modest restenosis,  $40\% \pm 9$  immediately post-PTCA to  $45\% \pm 15$  at baseline, among these lesions. For each post-PTCA lesion 13 non-PTCA lesions were selected, matched for lipid phenotype, LDLc, HDLc, and baseline stenosis severity. The severity of the 18 PTCA, and 202 non-PTCA lesions was measured in blinded fashion at baseline and 2.5 years later. LDLc and HDLc, essentially unchanged by CONV, changed substantially with N+C (-45%, +45%) and with L+C (-47%, +16%). Results are expressed as change ( $\pm$  SD) in % diameter stenosis between these two time points ( $\Delta\%S_{2.5}$ ), and frequency of lesion regression by at least 10% (RF).

	CONV				N+C				L+C			
	N	$\Delta\%S_{2.5}$	RE		N	$\Delta\%S_{2.5}$	RE		N	$\Delta\%S_{2.5}$	RE	
Non-PTCA Les.	40	$2 \pm 12$	5%		31	$3 \pm 6$	10%		131	$2 \pm 8$	12%	
PTCA Les.	6	$1 \pm 5$	0%		4	$-14^{*+} \pm 10$	$75\%^{*+}$		8	$-14^{*+} \pm 8$	$63\%^{*+}$	
chi-square comparison:	Versus non-PTCA:				$p < 0.05$				$p < 0.01$			
	Versus Conv:				$p < 0.05$							

**CONCLUSIONS:** Late regression of residual stenosis among PTCA lesions was significantly greater in frequency and magnitude than that among a matched set of non-PTCA lesions. This occurred only among patients taking intensive lipid-altering therapy. These findings suggest that such therapy 1) may be effective as primary therapy for significant but symptomatically stable restenosis, and 2) might retard restenosis.

10:45

Differences in the Progression of Coronary Artery Disease between the Three Major Coronary Arteries - Results from the INTACT-study

Stefan Jost, Wolf Raffienbeul, Jaap Deckers, Peter Nikutta, Birgitt Wiese, Paul R. Lichtlen, and the INTACT-Group. Hannover Medical School, Hannover, FRG

The progression (PRO) pattern in the three major coronary arteries was analyzed from the data of the prospective INTACT-study. In INTACT, 348 pts underwent repeated coronary angiography under standardized conditions within three years. The angiograms were quantitatively analyzed using a computer-assisted edge detection system (CAAS). A coronary stenosis (ST) was defined as diameter reduction  $\geq 20\%$ . A total of 4848 coronary segments and 1063 ST were compared between the angiograms. PRO of coronary artery disease was defined as reduction of the minimal diameter of preexisting ST by  $\geq 0.4$  mm (two-fold standard deviation of the method), as well as by development of new ST at previously normal sites. The incidence of PRO in the right coronary artery (RCA), circumflex (CX) and left anterior descending (LAD) artery is depicted in the table; A = ST with PRO; B = new ST/segment

	RCA	CX	LAD
A	115/420(27.4%)	74/313(23.6%)	64/330(19.4%)*
B	96/1546(6.2%)	73/1492(4.5%)	65/1810(3.8%)*

\* $p < 0.001$

Thus, PRO of preexisting ST, as well as the development of new ST was most frequent in the RCA. Revascularization measures should consider the particularly rapid PRO of the RCA.

11:00

QUANTITATIVE MEASUREMENTS OF APPARENTLY NORMAL CORONARY SEGMENTS IN PATIENTS WITH CORONARY ARTERY DISEASE

Wing-Hung Leung, Tommy C. Lee, Michael L. Stadius, Edwin L. Alderman, Stanford University, Stanford, CA

We evaluated angiographically normal coronary segments in patients (Pts) with visually evident coronary artery disease (CAD) elsewhere using quantitative coronary arteriography. We examined 136 angiograms of male Pts with CAD to identify visibly normal segments of the left main (LM) and proximal portions of the left anterior descending (pLAD), left circumflex (pLCX) and right coronary (pRCA) arteries. Normal segments were grouped according to the proximity of the CAD elsewhere. Normal segments without any visible disease in the same vessel, but with CAD present in other vessels, comprised the *Distant CAD* group. For the LM, this meant no disease in both the LAD and LCX vessels. Conversely, normal segments with visible disease elsewhere within the same vessel were considered to have *Adjacent CAD*. For the LM, this meant disease in the LAD and/or LCX. Normal segments with visibly abnormal adjacent segments comprised the *Immediately Adjacent CAD* group. Normal segments with visibly normal intervening segments between the normal-appearing proximal segment and distal disease comprised the *Adjacent but Distal CAD* group. Entirely normal angiograms from 26 age-matched males with atypical chest pain were used as controls. All segments were measured post-NTG using computer-assisted quantitation.

Mean Diameter (mm)	Control	n	Distant CAD	n	Adjacent but Distal CAD	n	Immediately Adjacent CAD	n
LM	4.48	21	4.35	5	$4.00^{* \ddagger}$	52	$3.75^{* \ddagger \S}$	14
pLAD	3.55	22	$3.12^{*}$	16	$3.00^{*}$	9	$2.54^{* \ddagger \S}$	12
pLCX	3.19	23	$2.95^{*}$	21	2.95	16	$2.44^{* \ddagger \S}$	21
pRCA	3.43	23	$3.22^{*}$	20	3.17	10	$2.68^{* \ddagger \S}$	16

\*  $p < 0.05$  vs Control;  $\ddagger$   $p < 0.05$  vs Distant CAD;  $\S$   $p < 0.05$  vs Adjacent but Distal CAD  
**Conclusions:** In CAD Pts, apparently normal coronary segments have smaller luminal diameters compared to similar segments in Pts with no evidence of CAD. These findings are consistent with the presence of diffuse CAD in the otherwise apparently normal segments from the CAD Pts. Quantitative angiographic techniques are useful in detecting abnormalities in coronary artery diameters which most likely reflect the presence of early, otherwise inapparent, CAD.